Case Reports

Severe Iron Deficiency Anemia

An Example of Covert Child Abuse (Münchausen by Proxy)

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THE EXPLOITATION AND ABUSE OF CHILDREN have been problems throughout history. Many accounts of corporal punishment and even murder of children appear in the Bible. In 1967 the battered child syndrome was first introduced in the medical literature by Kempe and co-workers.[1] Most of the reports on the battered child syndrome that followed their original article have concentrated on the physical abuse, sexual exploitation and neglect aspects of this disorder. Even Kempe and associates, however, in their original description alluded to the possibility of covert forms of child abuse in which a parent or parent substitute may assault a child by administering a drug overdose.

The term Münchausen syndrome was introduced by Asher in 1951 to describe patients with a syndrome of an addiction to hospital treatment.[2] (The original Baron von Münchausen was an 18th century German cavalry officer whose incredible, fictitious tales became famous.) Asher describes a group of patients who created fictitious symptoms and illnesses to gain admission to hospitals. In 1977 Meadows introduced the term "Münchausen syndrome by proxy" to describe situations in which one person's illness is induced or fabricated by someone else.[3] Since Meadow's original report, scattered case reports of Münchausen by proxy have appeared in the medical literature and have included deliberate poisoning of children,[4-6] parental insulin administration[7] and maternal fabrication of a child's illness.[3,8-12]

Our case differs slightly from these previously recorded cases of Münchausen by proxy. Instead of purposely giving her child a poison, the mother in our case chose to remove all available iron from her child's diet. As a result, a severe iron deficiency developed in the previously thriving child.

Report of a Case

The patient, a 14-month-old female infant, was admitted to Merced Community Medical Center for evaluation of severe hypochromic, microcytic anemia reportedly not responsive to iron therapy.

This child was born to a 19-year-old gravida 3, para 2, abortus 1 woman whose pregnancy was complicated by a very poor weight gain of only 2.27 kg (5 lb) during a term gestation. The mother was also noted to have anemia late in her third trimester, with a hematocrit of 26%. The mother's labor was complicated by a breech presentation and failure to progress, which resulted in a cesarean section delivery. The child was vigorous at birth with Apgar scores of 7 at one minute and 9 at five minutes. Her birth weight was 2.44 kg (5 lb 6 oz) with an estimated gestational age of 37 weeks, making the child somewhat small for the dates. The head circumference and length were appropriate for her age. The postnatal course was unremarkable, and the child was discharged home with the mother at 4 days of age.

She was readmitted at 2 weeks of age for an apparent apneic episode, associated with spitting up formula that was reportedly iron fortified. Evaluation of the patient showed no abnormalities and, on monitoring the child for several days, there were no other apneic spells. It was felt that overfeeding was the cause of her vomiting and subsequent apnea.

At 4 months of age, the child was seen for well-child care. During that visit, the mother stated her child was different from her other child and wondered if this child could hear. Review of child care during this visit included a confirmation of the use of iron-fortified formula. A hearing examination was also arranged. After several missed appointments, the child did receive a brain-stem auditory evoked-response test, the results of which were normal.

When she was 6 months old, the child was admitted to the hospital for fever and rectal bleeding. She was noted to have bilateral otitis media, rectal fissures and mild anemia, with a hemoglobin of 10 grams per dl. Review of her health care showed that she was following the 25th percentile in all of her growth measurements and was up to date on her immunizations.

The mother reported feeding her child formula with iron. The child was discharged home on an antibiotic regimen and therapeutic dosages of ferrous sulfate. During follow-up visits, the mother reported giving her child ferrous sulfate and iron-fortified formula and introducing iron-fortified cereals. The patient continued to grow normally, with resolution of the rectal fissures and rectal bleeding.

At 13 months of age, the child was seen in the emergency room for irritability and fever. Bilateral otitis media was diagnosed and she was noted to have a hematocrit of 23%. When seen in follow-up a few days later, the child was following her growth curves normally and her development was progressing without problems. The mother reported feeding her child a wide variety of table foods, including iron-fortified formula and cereal, and was still giving her child ferrous sulfate as described earlier. On closer questioning, the mother described the correct color can of formula for iron-fortified formula, documenting precisely how she was mixing the milk and how much her child was drinking. In addition, the mother reported in precise terms the amount of ferrous sulfate she was giving the child. She said that her child was not having any hematemesis, melena, blood per rectum or hematuria. A

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repeat hematocrit was 23%. The child's dose of ferrous sulfate was adjusted to her new weight and the mother was counseled once again regarding diet. A return appointment was given for one week later.

The mother missed her next appointment but did return three weeks later. She again reported the adequacy of the child's diet, could precisely recall how much therapeutic iron she was giving her child and denied any bleeding problems. A complete blood count done at this clinic showed a hemoglobin of 6.6 grams per dl and a hematocrit of 20% with microcytic and hypochromic indices.

It was decided to admit this child to hospital for further evaluation and also to investigate the home situation.

Laboratory evaluation elicited the following values: hematocrit 19.1%, hemoglobin 5.7 grams per dl, leukocyte count 12,600 per μl (26% polymorphonuclear leukocytes, 1% bands, 69% lymphocytes and 4% monocytes); erythrocyte count 235,000 per μl, mean corpuscular volume 49 cu microns, mean corpuscular hemoglobin 14 pg, a peripheral smear showing microcytic and hypochromic erythrocytes, serum ferritin less than 1 ng per ml (normal 7 to 140), serum iron 14 μg per dl (normal 30 to 70), total iron binding capacity 411 μg per dl (normal 250 to 400), percent saturation 3.5% (normal 10% to 50%); results of hemoglobin electrophoresis, urinalysis and electrolytes were normal; blood urea nitrogen 9 mg per dl and creatinine 0.3 mg per dl. A diagnosis of severe iron deficiency anemia was made and therapeutic dosages of ferrous sulfate were started.

Seven days after therapy was initiated in the hospital, the hematocrit had risen to 31% with a reticulocyte count of 4.8%. Stool guaiacs were negative throughout her hospital stay. The child was noted to be developmentally normal, although the only words she was heard to utter were "shut up."

Investigation of the home revealed domestic turmoil. The mother had recently separated from her husband and moved out of the house with her two children. When told that her child's anemia was most likely the result of dietary deficiency of iron and noncompliance with iron therapy, the mother's affect remained flat. She continued to maintain that she diligently provided her child with sources of dietary and supplemental iron. The mother did, however, admit that her child had always been a source of trouble, even during pregnancy, and that this child was different from her older one.

After a seven-day stay in hospital, the child was discharged home to the mother on a regimen of appropriate doses of liquid ferrous sulfate. Follow-up with Child Protective Services and a public health nurse was arranged to help assure compliance, and the child's anemia was completely corrected. The mother was referred to mental health services for counselling. The father, however, gained custody of the children two months after hospitalization when the mother failed both mental health and pediatric clinic appointments.

Following this, the child had one other admission to hospital for a simple febrile seizure at 18 months of age. Otherwise, the patient continued to do well and to have normal blood counts.

Discussion

This is an unusual case that shows how a mother elected to remove all available iron from her child's diet, yet provided an adequate diet and environment to ensure normal growth and development. This type of illness, one that is induced by another, could be classified as covert child abuse or Münchausen by proxy.[13] Anemia is a common childhood ailment that can easily be diagnosed by dietary history and response to iron therapy.[14] The correct diagnosis in this case, as in other forms of covert child abuse, is frequently delayed because of a misleading history, delay in seeking medical attention and failure to comply with medical advice. In cases of unusual signs, symptoms or illness in children, one should always consider covert child abuse in the differential diagnosis.

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Metoclopramide-Induced Reversible Impotence

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METOCLOPRAMIDE, a drug used to enhance gastrointestinal motility and to control nausea, is known to be a central dopamine-receptor blocker and by that mechanism to lead to hyperprolactinemia. The side effects of metoclopramide are mainly central nervous system-mediated effects such as somnolence, extrapyramidal reactions and agitation. In this report, I describe two cases of reversible metoclopramide-induced impotence and discuss the implications of this association in view of the known pharmacology of the drug.

Reports of Cases

Case 1

The patient, a 62-year-old man, was admitted to hospital for recurrent nausea and vomiting. The patient had a 16-year history of ulcer disease and 5 years earlier had an antrectomy

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